Eliminating the trigger for polymorphic ventricular tachycardia

Molitor, N ; Breitenstein, A ; Saguner, A M

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Radiofrequency catheter ablation after unsuccessful treatment with an ICD

Eliminating the trigger for polymorphic ventricular tachycardia

Nadina Molitor, Alexander Breitenstein, Ardan M.Saguner

Department of Cardiology, University Heart Centre Zurich, Switzerland

Summary

A 60-year-old female patient was referred to our hospital because of recurrent syncope. The resting 12-lead ECG showed sinus rhythm with left bundle branch block and a high burden of monomorphic single premature ventricular beats (PVCs). Imaging studies confirmed a structurally normal heart and ischaemic heart disease was ruled out by coronary angiography. Telemetry revealed repeated episodes of polymorphic ventricular tachycardia (VT) triggered by monomorphic PVCs causing syncope. Therefore, an ICD was implanted and the patient was discharged on bisoprolol 10 mg/day. A few weeks later, she was referred to our hospital owing to repeated ICD shocks triggered by monomorphic PVCs with long-short sequences. Radiofrequency catheter ablation targeting the monomorphic PVC causing polymorphic VT was performed. With an almost perfect pace map within the distal coronary sinus, ablation in this region did not abolish the PVC. Therefore, we antegradely mapped the LV via transseptal access. Endocardial mapping at the anterior mitral anulus using the reverse S-curve technique revealed the earliest bipolar activation in close proximity to the ablated region within the coronary sinus. Ablation at this site abolished the clinical PVC. ECG monitoring at 3, 6 and 12 months did not reveal any PVC, and no ICD discharges have occurred since ablation.

Key words: polymorphic ventricular tachycardia; ventricular fibrillation; radiofrequency ablation

Clinical case

A 60-year-old female patient was referred to our hospital with a worsening history of palpitations and recurrent unprovoked syncopes. She denied dyspnoea and chest pain. Her medical history was remarkable for mild arterial hypertension, dyslipidaemia and depression, well controlled by her current medication, which she had taken for many years (atorvastatin, losartan, acetyl salicylic acid, fluoxetine). Family history was negative for cardiomyopathies/channelopathies and premature sudden cardiac death.

On clinical examination the patient appeared in good general condition. She was normotensive; heart and lung auscultation were normal without any sign of heart failure. Except for a mildly elevated C-reactive protein (47 mg/l) of unknown origin, laboratory values including serum potassium and magnesium were within normal limits. The resting 12-lead ECG showed sinus rhythm with left bundle branch block and a high burden of monomorphic premature ventricular beats (PVCs; fig. 1A). A transthoracic echocardiogram (TTE) and cardiac magnetic resonance imaging (MRI) ruled out structural heart disease, and absence of relevant ischaemic heart disease was confirmed by coronary angiography. Telemetry revealed repeated episodes of polymorphic ventricular tachycardia (VT) triggered by a monomorphic PVC (fig. 1B) causing (pre)syncope. Because of sustained polymorphic VT and syncope, the patient underwent implantation of a secondary prevention subcutaneous implantable cardioverter defibrillator (S-ICD; a structurally normal heart, only polymorphic VT without an indication for ATP and no bradycardia pacing indication) and was discharged on bisoprolol 10 mg/day.

In the following weeks, she was referred to our hospital several times because of repeated ICD shocks, which were always triggered by monomorphic PVCs with long-short R-R sequences prior to polymorphic VT. A 24-hour Holter ECG revealed 34 000 monomorphic PVCs. Therefore, we decided to perform radiofrequency catheter ablation targeting the monomorphic PVC that potentially triggered polymorphic VT. Since the origin of the PVCs was suspected to be close to the anterior part of the mitral annulus of the left ventricle (LV; right bundle branch block morphology, positive QRS in V1–V6, inferior axis, notched and broad QRS), we first mapped the distal coronary sinus with a steerable unidirectional (D-curve) 7.5 F Navistar ThermoCool ablation catheter using CARTO as an electroanatomical mapping system (Biosense Webster, Diamond Bar, CA). A good pace map (QRS match in 11 out of 12 leads) was noted in the suspect region within the distal coronary sinus (fig. 2A, coronary sinus in grey), suggesting an epicardial/intramural origin of the PVCs. However, ablation within this region with 20–25 W (fig. 2A, pink dots in the coronary sinus) did not abolish the PVCs. Therefore, we performed a single anteroinferior transseptal puncture guided by fluoroscopy with a BRK-1 needle and a 8.5F SL1 sheath (Abbott, formerly St. Jude Medical, St. Paul, MN), and antegradely mapped the endocardial site of the LV (fig. 2A, LV in green). Endocardial mapping in the LV at the anterior mitral annulus using the reverse S-curve technique de-

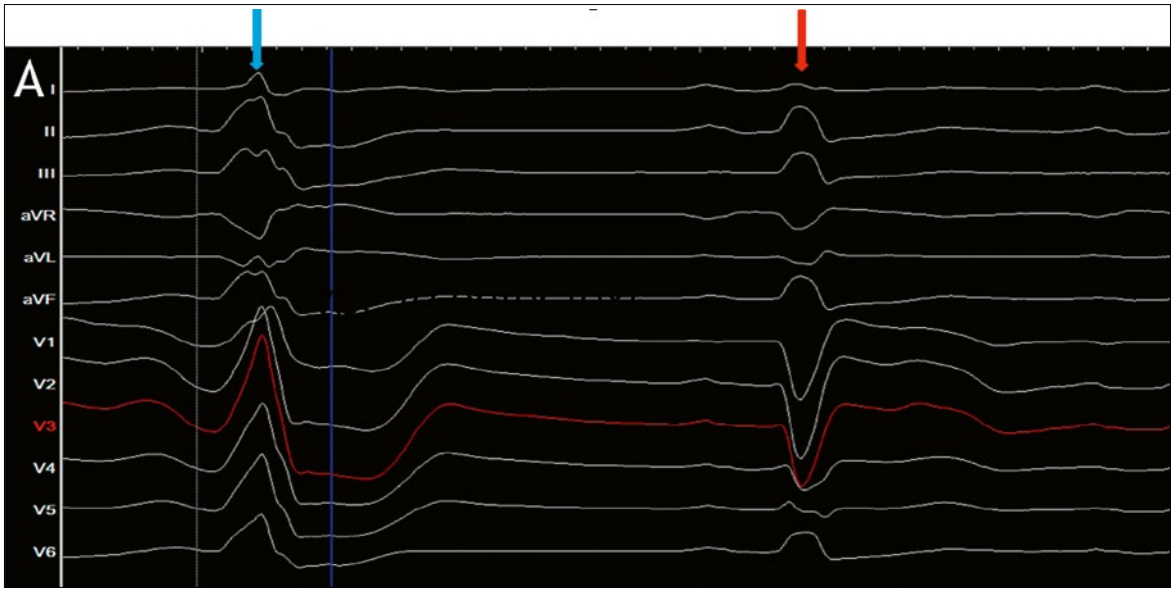

Figure 1A: Clinical monomorphic PVC (blue arrow, sweep speed 100 mm/s) that was targeted with catheter ablation. The intrinsic QRS complex during sinus rhythm (red arrow) shows complete left bundle branch block.

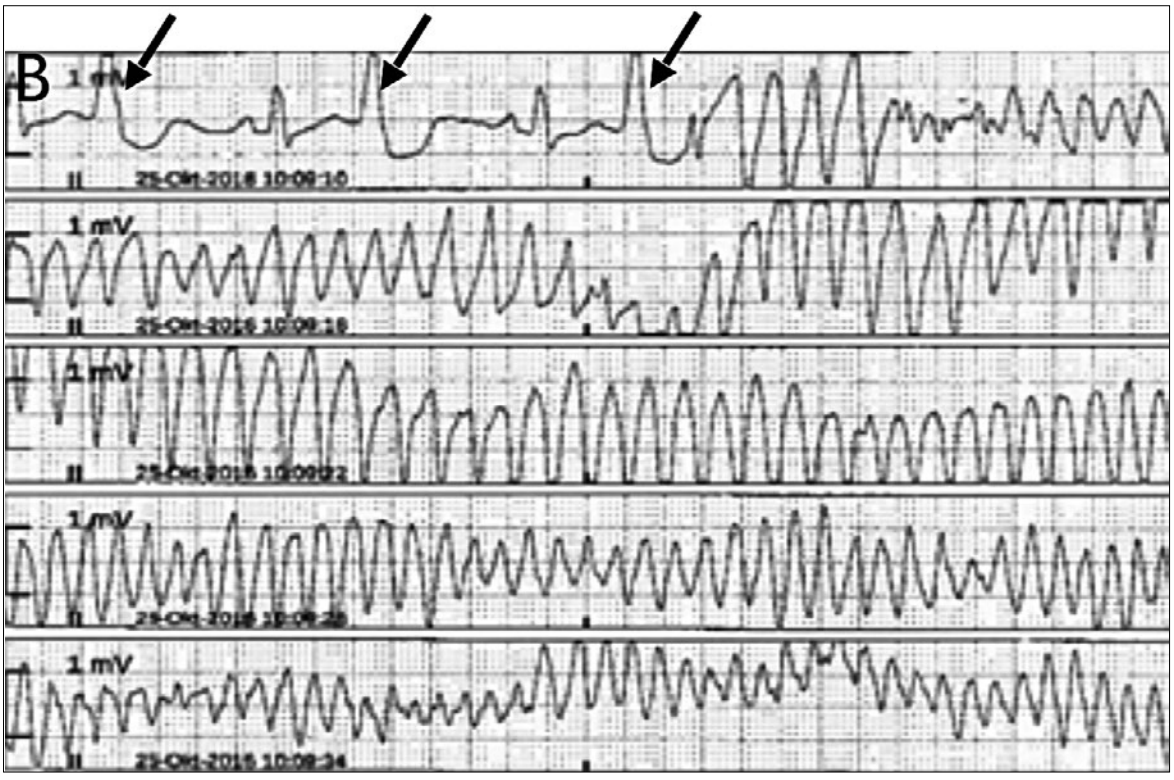

Figure 1B: Clinical PVCs (black arrows, sweep speed 25mm/s) leading to long-short R-R sequences and induction of polymorphic VT during telemetry monitoring. Polymorphic VT correlated with clinical symptoms of the patient.

scribed by F. Ouyang et al. in 2014 ([1]; fig. 2B) revealed, in close proximity to the ablated region in the distal coronary sinus (fig. 2A, pink dots), the earliest bipolar local activation (20 ms) on the ablation catheter during PVC as compared with the onset of the QRS complex in the 12-lead surface ECG and a QS pattern on the unipolar electrogram (fig. 3A). Ablation at this site (fig. 2A, purple point) abolished the clinical PVC after 2 seconds (fig. 3B). The area was ablated with 30–35 W for 90 seconds in total. The periprocedural course was uneventful. ECG and Holter monitoring at 3, 6 and 12 months did not reveal any PVC, and no ICD discharges have occurred since ablation. The patient has been free from any cardiovascular symptoms since then.

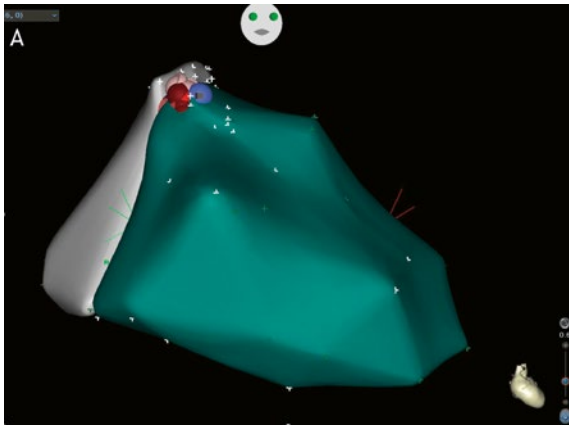

Figure 2A: Electroanatomical endocardial mapping of the left ventricle (green, a.p. view) and coronary sinus (grey) with CARTO3. Pink and red dots indicate ablation points from within the distal coronary sinus and endocardial anterior mitral annulus. The purple point indicates the site of successful elimination of the PVC.

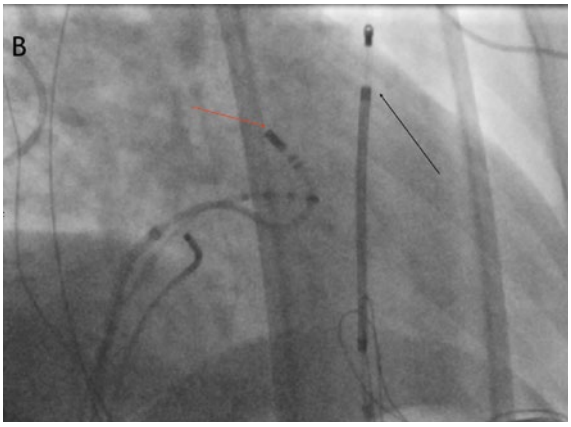

Figure 2B: Fluoroscopic image of catheter positions during successful ablation of the PVC from the endocardial LV at the anterior mitral annulus. The red arrow indicates the tip of the ablation catheter using the reverse S-curve technique described by F. Ouyang et al., the black arrow indicates the defibrillation lead of the subcutaneous ICD at the left parasternal border (RAO 30°).

Conclusions

Idiopathic polymorphic VT / ventricular fibrillation (VF) can occur in structurally normal hearts and comprises various clinical entities [2]. In particular, PVCs have been reported to provoke idiopathic polymorphic VT/VF [3, 4]. In our case, we also assumed an idiopathic origin of the clinical PVC. However, we cannot exclude fibrosis on a microscopic level as a cause of PVC with recurrent polymorphic VT, since the resolution of MRI, TTE and electroanatomical voltage mapping is not high enough to visualise small areas of fibrosis. T1 mapping by MRI can be a better method to search for smaller areas of fibrosis, but was not available in our case. Furthermore, we created the LV map as an activation map during PVCs. Indeed, this showed some borderline low voltage areas in the region of interest prior to ablation, but reference voltage map cut-off values in prior studies have been de-

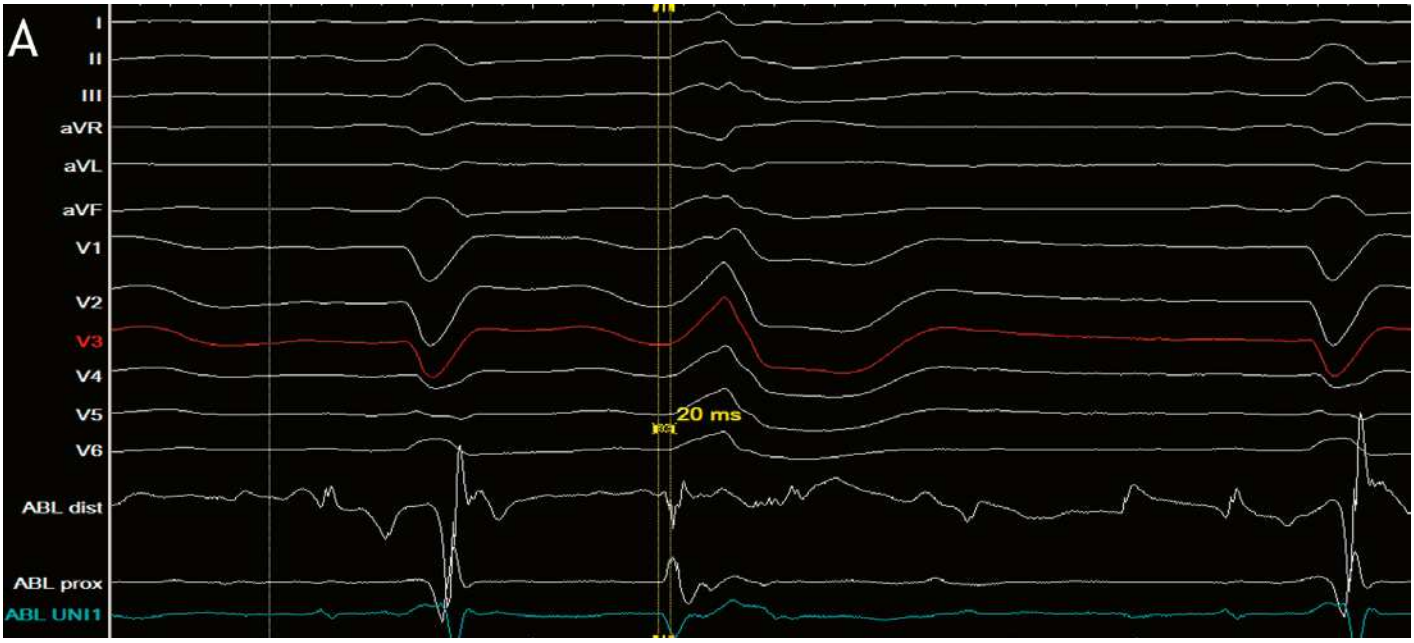

Figure 3A: Tracing (sweep speed 100 mm/s) indicates the earliest local bipolar activation on the ablation catheter during endocardial mapping in the LV at the anterior mitral annulus (20 ms before the onset of the QRS complex during the PVC in the 12-lead ECG). Please note also the QS complex on the unipolar electrogram. Leads I, II, III, aVR, aVL, aVF, V1-V6 indicate 12-lead surface ECG. ABL dist, prox and UNI indicate distal and proximal bipolar as well as the unipolar signal on the ablation catheter.

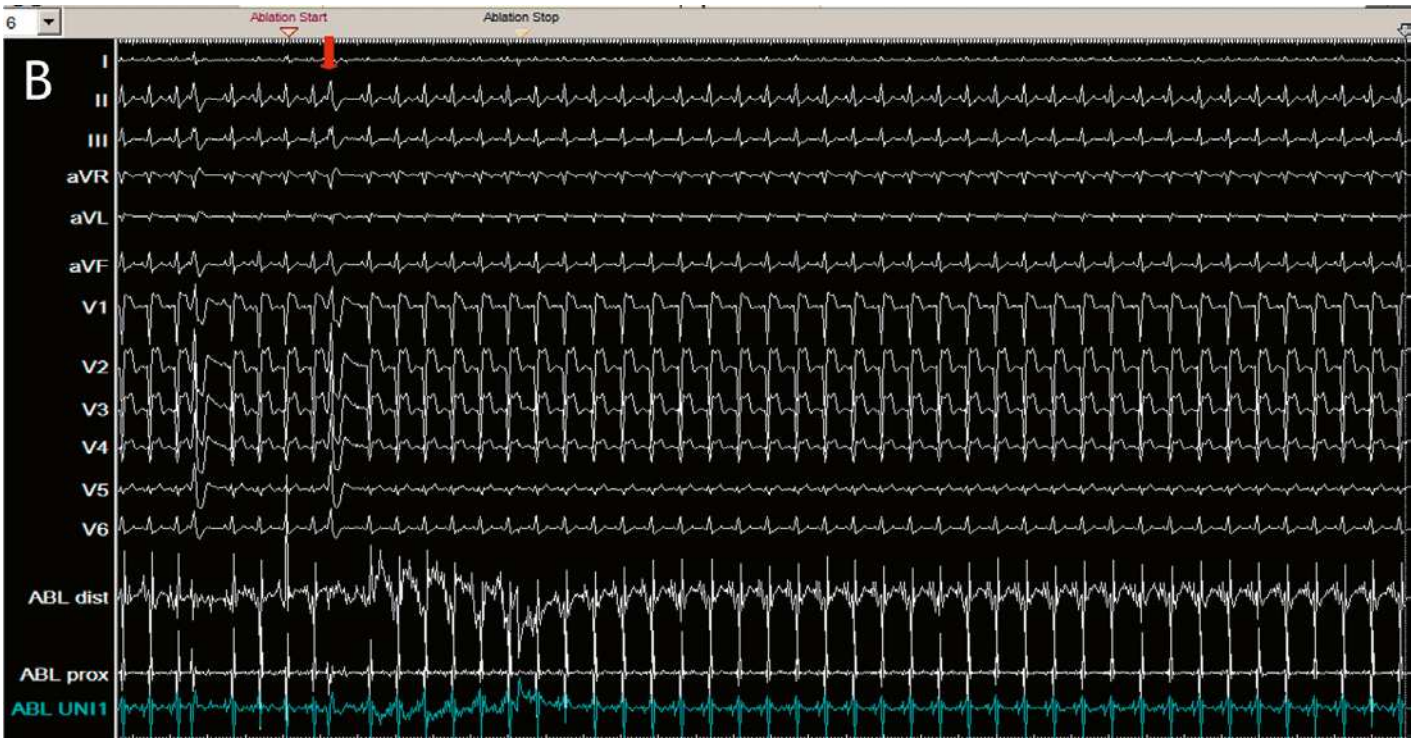

Figure 3B: Tracing (sweep speed 6mm/s) shows successful elimination of the clinical PVC two seconds after the onset of radiofrequency energy delivery (red arrow). Please note that due to ablation in this region and changes in the coupling interval the PVC morphology slightly changed during the procedure. Leads I, II, III, aVR, aVL, aVF, V1-V6 indicate 12-lead surface ECG. ABL dist, prox and UNI indicate distal and proximal bipolar as well as the unipolar signal on the ablation catheter.

termined during sinus rhythm, therefore voltage mapping during PVCs may not be reliable.

Catheter ablation for the treatment of recurrent PVC-triggered polymorphic VT/VF was first described by Haissaguerre et al. in 2002 in a group of patients with structurally normal hearts [5]. The idea was that polymorphic VT/VF is "triggered" by short coupled PVCs falling into the "vulnerable period' of the QRS complex, leading to an R-on-T phenomenon, and consequently polymorphic VT or VF. Since the initial report, the feasibility of eliminating polymorphic VT/VF by targeting PVC triggers has been reported in a number of clinical situations, including patients with idiopathic VF [6] and structural heart disease [7, 8, 9]. Most published series on VF ablation have reported immediate success rates ranging from 81 to 100% [5, 6, 10]. However, a long-term follow-up study reported a recurrence rate of VF of 18% after a median of 24 months [9]. Therefore, the concept of catheter ablation of monomorphic PVCs triggering polymorphic VT/VF as a substitute for ICD therapy warrants further investigation.

Correspondence:
Ardan M. Saguner, MD
Department of Cardiology
University Heart Center
Zurich
Rämistrasse 100
CH-8091 Zurich
Ardansaguner[at]yahoo.de

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